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Health and Environmental Effects Profile for Ethoprop

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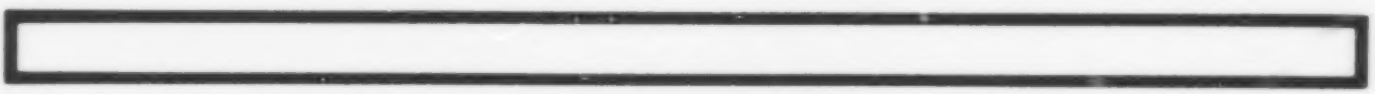

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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR ETHOPROP

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16. ABSTRACT

The Health and Environmental Effects Profile for ethoprop was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates have been presented provided sufficient data are available. Existing data are insufficient to determine an Acceptable Daily Intake (ADI) or a carcinogenic potency factor for ethoprop.

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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents.

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include, Acceptable Daily Intakes (ADIs) for chronic exposures. An ADI is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, ADIs are not estimated in this document series. Instead, a carcinogenic potency factor or q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA docket.

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LIST OF ABBREVIATIONS

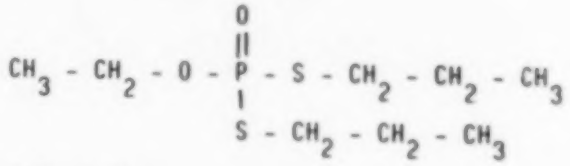
ADI	Acceptable daily intake
BCF	Bioconcentration factor
EC_{50}	Median effective concentration
LC_{50}	Concentration lethal to 50% of recipients
LD_{50}	Dose lethal to 50% of recipients
NOAEL	No-observed-adverse-effect-level
ppm	Parts per million

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

Ethoprop is also known as Mocap, Ethoprophos, Jolt, Prophos, VC9-10, O-ethyl-S,S-dipropyl-phosphorodithioate and the O-ethyl-S,S-dipropyl ester of phosphorodithioic acid [British Crop Protection Council (BCPC), 1977].

Its chemical structure is given below:



Molecular formula: $C_8H_{19}O_2PS_2$

Molecular weight: 242.36

The Chemical Abstracts Service (CAS) Registry number for this compound is 13194-48-4.

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Ethoprop is a clear, pale yellow liquid under ambient conditions. Some of its physical properties are given below (BCPC, 1977):

Boiling point at 0.2 torr:	86-91°C
Water solubility at 25°C:	750 mg/ℓ
Density:	1.094 g/cm³ at 20°C
Vapor pressure:	3.5×10^{-4} mm of Hg at 26°C

1.3. PRODUCTION DATA

Ethoprop is produced by reacting ethyl phosphorodichloridate and 1-propanethiol in the presence of aqueous sodium hydroxide (BCPC, 1977). Ethoprop was produced in 1982 by Rhone-Poulenc Inc., in Mount Pleasant, TN, under the trade name Mocap® [Stanford Research Institute (SRI), 1983]. Production data for 1982 are unavailable, but 9 million pounds were produced in 1976 (Ayers and Johnson, 1976).

1.4. USE DATA

Ethoprop is a phosphorodithioate pesticide used to control nematodes, wire-worms, fleabeetle larvae, antworm and corn rootworm on many food crops, tobacco and commercial turf grasses. It is most frequently applied by soil incorporation at rates of 1.6-6.6 kg/ha (BCPC, 1977).

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

Few data regarding the fate of ethoprop in soil and water and no data regarding its fate in the atmosphere were located in the available literature.

Ethoprop does not undergo hydrolysis in aqueous solution at ambient temperature and low pH. Rapid hydrolysis occurs in basic aqueous solutions at 25°C. Its half-life in freshwater at pH 11-12 at 25°C is 26 minutes (U.S. EPA-NIH, 1983). Similarly, Menzer et al. (1971) reported that the half-life of ethoprop in 0.05 N NaOH was 35 minutes at 25°C, and very little hydrolysis occurred in 0.1 N HCl during a 60-minute incubation at the same temperature. Kenaga (1980) calculated a bioconcentration factor of 15 for ethoprop, based on its water solubility. This low value indicates that the compound will not bioconcentrate to a great degree.

The persistence of ethoprop in soil has been evaluated by bioassay by several investigators. Hamlen et al. (1979) applied ethoprop (22.4 kg/ha) to porous and dense peat soils, and observed its effect on Diaprepes abbreviatus, a sugarcane rootstalk borer weevil. Ethoprop was effective initially (18 hours postapplication), but showed a lack of residual activity at 6 weeks and no activity after 3 months of weathering. Ethoprop was observed to be slightly more persistent in the dense soil. Argauer and Cantelo (1980) studied the stability of ethoprop in mushroom compost by gas chromatograph and bioassay. Ethoprop incorporated into mushroom compost (25 ppm) decreased to <1 ppm in 50 days and showed a loss of potential to control sciarid flies, Lycoriella mali, after 20 days under mushroom growing conditions.

The seasonal movement and fate of ethoprop in soil have been experimentally determined and compared to computer models based on physicochemical

data (Smelt et al., 1981; Leistra and Smelt, 1981; Leistra, 1979). Leistra (1979) measured the soil adsorption coefficients of ethoprop (0.95 and 3.64 ppm solution) applied to columns of soils that varied within specified limits of organic matter content, clay, silt and pH values. Adsorption coefficients ranged from 0.74×10^{-3} to 7.4×10^{-3} mmol kg^{-1} (soil)/mmol m^{-3} (solution). Higher adsorption coefficients were associated with soils having higher values for organic matter content, clay and silt, and lower pH. Higher ethoprop concentrations were associated with lower soil adsorption. Computer simulated concentration profiles using spring rainfall data indicated that ethoprop did not penetrate lower than 0.2 m up to 60 days. A simulated applicated rate of 10 kg ethoprop/ha at a depth of 0.1 m indicated that <10% of the applied dose volatilized in 60 days (Leister, 1979). Similar research was conducted to evaluate the fate of ethoprop in soil during winter months (Smelt et al., 1981; Leistra and Smelt, 1981). Cores of silt loam, sandy loam and humous sandy loam were characterized, replaced in the field and treated with ethoprop (10.3 kg/ha). Weather conditions and water elution patterns were recorded throughout winter exposure of the soil columns. Concentration peaks were observed at a depth of 15-20 cm, but ethoprop was not detected below 30 cm or in column effluent. The ranges of half-life values observed in incubated silty loam (12 days at 20°C to 89 days at 2°C), sandy loam (16 days at 20°C to 144 days at 2°C) and humous loamy sand (73 days at 20°C to 347 days at 6°C) indicated that soil composition and temperature influerce the rate of conversion (Smelt et al., 1981). A computer model based on physicochemical data for ethoprop allowed fair predictability of ethoprop movement (Leistra and Smelt, 1981).

Rohde et al. (1979) studied the effects of granular and liquid ethoprop formulations on its decomposition and presence in subsurface and runoff

water after application. Granular or liquid ethoprop was applied (9 kg/ha) at a depth of 15 cm in Cowarts sandy loam by incorporation or injection, respectively. Two days after application, the top 10 cm of soil treated with granular and liquid ethoprop contained 710 ng/g and 1207 ng/g, respectively. Runoff water 2-3 days after application of granular and liquid ethoprop contained 45 and 283 µg/ℓ, respectively. Approximately 0.1 and 0.01% of the liquid ethoprop applied was found in the surface and subsurface runoff, respectively. Surface water removed 0.01% of the granular dose, but subsurface flow did not occur. Over 90% of the ethoprop contained in either formulation decomposed in the soil by 30 days after application.

Tu (1973) found that the addition of ethoprop to soil at 5 or 30 µg/g soil stimulated microbial oxygen consumption and that the stimulation was directly proportional to the concentration of ethoprop. The degree of stimulation was greater at 30°C than at 15°C and was greater in the presence of glucose than in its absence. Menzer et al. (1971) reported that no metabolism of ethoprop was observed in autoclaved soil. These results suggest that microbial degradation may contribute significantly to the disappearance of ethoprop from soil.

3. EXPOSURE

Argauer and Feldmesser (1978) reported detecting ethoprop residues in several vegetables grown in soil treated 1 week before planting with 3.4, 6.7 or 13.4 kg ethoprop/ha. Residues were not detected (<0.01 ppm) in whole beets, cabbages, cantaloupe, peas and tomatoes. Ethoprop residues were detected in whole onions (0.12-1.34 ppm), radishes (0.12-0.66 ppm), carrots (0.14-0.81 ppm) and eggplant (0.027-0.086 ppm). Select parts of certain vegetables ranged from 0.02 ppm in beet skin to 20 ppm in carrot side root hairs. Increasing residue concentrations were associated with increased application rate.

Narain et al. (1980) reported ethoprop residues in vegetables grown in a home garden in Virginia. The soil was treated with 0, 3.4, 6.7 or 13.4 kg ethoprop/ha, and vegetables were planted 1 week later. Ethoprop residues were detected in carrots (0.06-0.78 ppm), peas (0.01-2.12 ppm), lima beans (0.02-4.16 ppm), radishes (0.01-0.14 ppm), string beans (0.01-0.11 ppm) and turnips (0.01-0.15 ppm). Residues were generally higher in vegetables grown in soil treated with 6.7 kg/ha than with other application rates.

Hunt et al. (1981) reported ethoprop residues on vegetables grown in soil treated with 0, 3.4, 6.7 or 13.4 kg/ha. Vegetables were planted 4 or 40 days after ethoprop application. Ethoprop residues were not detected (<0.005 ppm) in tomatoes, cucumbers or lettuce. Residues were detected in turnip roots (<0.005-0.018 ppm), onions (<0.005-0.068 ppm) and radishes (0.005-0.345 ppm). Increased residue concentrations were associated with increased application rates.

4. PHARMACOKINETICS

4.1. ABSORPTION

The acute lethal toxicity of ethoprop after dermal application (dermal LD_{50} for rabbit = 26 mg/kg) demonstrates that it is well absorbed by this route (BCPC, 1977).

Iqbal and Menzer (1972) demonstrated rapid absorption of ethoprop upon oral administration to rats. Rats excreted ~50% of radioactivity contained in a single gavage dose of ^{14}C-ethyl ethoprop (dissolved in 0.25 mℓ or distilled water) in the urine within 6 hours.

Pertinent data regarding the pulmonary absorption of ethoprop were not located in the available literature.

4.2. DISTRIBUTION

Pertinent data regarding the distribution of ethoprop were not located in the available literature.

4.3. METABOLISM

Iqbal and Menzer (1972) studied the in vivo and in vitro metabolism of ^{14}C-labeled ethoprop by rats and rat liver microsomes. To characterize the degradation of ethoprop, 3 male and 3 female SD rats were given single gavage doses containing 1.28 x 10^6 cpm/rat (1.25 m Ci/mmole) of ethyl-^{14}C and 3.0 x 10^6 cpm/rat (2.8 m Ci/mmole) of propyl-^{14}C labeled ethoprop. The compounds were dissolved in 0.25 mℓ of distilled water. Rats were placed in metabolism cages and urine was collected 6, 12, 24, 36, 48 and 72 hours after administration of the ethyl-^{14}C labeled compound and 8, 24, 48 and 72 hours after administration of the propyl-^{14}C labeled compound. Feces were collected for 72 hours after administration. The urine was extracted with chloroform; urinary metabolites in the chloroform

and water fractions were identified by column and thin-layer cochromatography and were assayed for radioactivity. No radioactivity was found in the chloroform extract of urine from rats given the ethyl-^{14}C labeled compound. Five metabolites were found in the water fraction and accounted for ~50 and 58% of the administered dose after 72 hours in male and female rats, respectively. The metabolites included O-ethyl phosphoric acid, O-ethyl-S-propyl phosphorothioic acid (major metabolite) and 4 unknown hydrolytic products. Three metabolites, methyl propyl sulfide, methyl propyl sulfoxide and methyl propyl sulfone, were found in the chloroform extract of urine from rats given propyl-^{14}C ethoprop and accounted for ~2% of the administered dose. The water fraction of urine from these rats contained 3 unknowns and S-propyl phosphorothiolic acid, O-ethyl S-propyl phosphorothioic acid (major metabolites) and S,S-dipropyl phosphorodithioic acid, and represented 65 and 66% of its administered dose in male and female rats, respectively. Less than 1% of the administered dose was found in the 72-hour fecal samples from rats given the ethyl-^{14}C and propyl-^{14}C labeled compounds.

The authors proposed a metabolic pathway for ethoprop that involves deethylation and depropylation, formation of propyl thiolate ions, and subsequent steps including oxidation, further dealkylation and conjugation with glutathione. Rats and rabbits pretreated with sodium phenobarbital yielded liver microsomes that produced greater quantities of metabolites in in vitro assays than did microsomes from untreated rats. The specific metabolites were similar to those identified in in vivo assays.

4.4. EXCRETION

Rapid metabolism and excretion (primarily urinary) of ethoprop was observed in rats given gavage doses of ethyl or propyl-^{14}C labeled

ethoprop (Iqbal and Menzer, 1972). Within 72 hours of dosing, urinary excretion accounted for ~50% of the radioactivity from the ethyl-labeled compound and ~65% of the radioactivity from the propyl-labeled compound; fecal excretion accounted for <1% of the administered radioactivity regardless of label position. The fate of the rest of the administered radioactivity was not determined. Neither the expired air nor the carcass was monitored for radioactivity.

5. EFFECTS

5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of ethoprop were not located in the available literature. Ethoprop is not scheduled for testing by the NTP (1983) and because of the absence of experimental data has not been considered by the [International Agency for Research on Cancer (IARC), 1982].

5.2. MUTAGENICITY

Gentile et al. (1982) studied the mutagenicity of a commercial grade of ethoprop in Salmonella typhimurium and Zea mays. Ethoprop did not induce reverse mutation in S. typhimurium in plate incorporation assays with or without rat or Z. mays activation (dose not specified). In an in situ assay, ethoprop was applied to soil at 2.24 kg/ha before emergence of Z. mays. Plants were allowed to grow for 12-14 weeks, and after this growth period pollen grains were evaluated for mutations at the waxy locus by observing staining patterns with iodine. A significant ($P<0.05$) increase in reversions was observed when compared with controls.

5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity of ethoprop were not located in the available literature.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding other reproductive effects of ethoprop in mammals were not located in the available literature.

5.5. SUBCHRONIC AND CHRONIC TOXICITY

According to (BCPC, 1977), dogs and rats "fed a diet containing 100 ppm ethoprophos for 90 days showed depression of cholinesterase levels but no other effects on pathology or histology." No other details were reported

and the source of these data was not mentioned. No other studies of the subchronic or chronic toxicity of ethoprop to mammals were located in the available literature.

5.6. OTHER RELEVANT INFORMATION

Acute exposure to ethoprop can result in symptoms typical of cholinesterase inhibitors. These include abnormal heart rate, convulsions, depression of the circulatory and respiratory systems and death by asphyxia because of respiratory failure (U.S. EPA-NIH, 1983).

The [National Institute for Occupational Safety and Health (NIOSH), 1983] reports LD_{50}s for oral administration in rats (34 mg/kg) and skin administration in rats (60 mg/kg) and rabbits (26 mg/kg). The BCPC (1977) reports an oral LD_{50} for rats of 62 mg/kg.

When assayed *in vitro* for inhibition of acetylcholinestase, ethoprop was relatively inactive. After incubation of ethoprop with a hepatic microsomal mixed-function oxidase, the resulting metabolite (probably the sulfoxide) was highly inhibitory to acetylcholinesterase (Wing et al., 1983).

6. AQUATIC TOXICITY

6.1. ACUTE TOXICITY

The 96-hour static LC_{50} for ethoprop (92% purity) in rainbow trout, Salmo gairdneri, was 1.15 mg/ℓ (Table 6-1) (U.S. EPA, 1975).

Acute static toxicity testing of ethoprop at 0.032, 0.056, 0.100, 0.180 and 0.320 mg/ℓ was conducted with marine spot, Leiostomus xanthurus, for exposures of 24, 48, 72 and 96 hours [Environmental Research Laboratory, (ERL), 1981]. At 0.056 mg/ℓ, 50% mortality was reported for 24- and 48-hour exposures; mortality was 83% at 72 and 96 hours (Table 6-1). A concentration of 0.032 mg ethoprop/ℓ for 96 hours produced 50% mortality and the 96-hour LC_{50} determined by probit analysis was 0.033 mg/ℓ. The sheepshead minnow, Cyprinodon variegatus, was less sensitive to ethoprop than spot during static toxicity testing. The mortality among groups of this species exposed to 1.0 mg ethoprop/ℓ for 24, 48, 72 and 96 hours was 30, 55, 60 and 65%, respectively. The 96-hour static LC_{50} was determined to be 0.74 mg/ℓ. Acute flow-through toxicity testing was done with sheepshead minnows exposed to ethoprop for comparison (ERL, 1981). Mortality after 24, 48, 72 and 96 hours exposure at 0.600 mg ethoprop/ℓ was 20, 80, 95 and 95%, respectively. The 96-hour LC_{50} determined by probit analysis in the flow-through system was 0.180 mg/ℓ. Mortality of pinfish, Lagodon rhomboides, in flow-through toxicity tests was 15, 65, 70 and 75% in fish exposed to 0.0077 mg ethoprop/ℓ for 24, 48, 72 and 96 hours, respectively (ERL, 1981). The 96-hour flow-through LC_{50} was determined to be 0.0063 mg ethoprop/ℓ (Table 6-1).

Toxicity testing of ethoprop has been conducted in several species of marine invertebrates (ERL, 1981). Mortality in the nysid shrimp, Mysidopsis bahia, was 0, 15, 60 and 80% in groups exposed to 0.032 mg ethoprop/ℓ for

TABLE 6-1

Acute Lethal Effects of Ethoprop to Marine Fish

Species	Duration (hour)	Concentration (mg/ℓ)	Method	Effect	Reference
Rainbow trout, *Salmo gairdneri*	96	1.15	static	LC_{50}	U.S. EPA, 1975
Spot, *Leiostomus xanthurus*	24	0.056	static	50% mortality	ERL, 1981
	48	0.056	static	50% mortality	ERL, 1981
	72	0.056	static	83% mortality	ERL, 1981
	96	0.056	static	83% mortality	ERL, 1981
	96	0.033	static	LC_{50}	ERL, 1981
Sheepshead minnow, *Cyprinodon variegatus*	24	1.00	static	30% mortality	ERL, 1981
	48	1.00	static	55% mortality	ERL, 1981
	72	1.00	static	60% mortality	ERL, 1981
	96	1.00	static	65% mortality	ERL, 1981
	96	0.74	static	LC_{50}	ERL, 1981
Sheepshead minnow, *C. variegatus*	24	0.60	flow-through	20% mortality	ERL, 1981
	48	0.60	flow-through	80% mortality	ERL, 1981
	72	0.60	flow-through	95% mortality	ERL, 1981
	96	0.60	flow-through	95% mortality	ERL, 1981
	96	0.18	flow-through	LC_{50}	ERL, 1981
Pinfish, *Lagodon rhomboides*	24	0.0077	flow-through	15% mortality	ERL, 1981
	48	0.0077	flow-through	65% mortality	ERL, 1981
	72	0.0077	flow-through	70% mortality	ERL, 1981
	96	0.0077	flow-through	75% mortality	ERL, 1981
	96	0.0063	flow-through	LC_{50}	ERL, 1981

24, 48, 72 and 96 hours, respectively. The penaeid shrimp, Penaeus stylirostris, was more sensitive to ethoprop, having 8, 45, 60 and 90% mortality during 24-, 48-, 72- and 96-hour exposures at 0.010 mg/ℓ. The 96-hour static LC_{50} values for M. bahia and P. stylirostris were 0.0230 and 0.0064 mg ethoprop/ℓ, respectively (ERL, 1981). In flow-through toxicity tests the 96-hour LC_{50} value for M. bahia was 0.0075 mg/ℓ. Flow-through testing was used to determine a 96-hour LC_{50} value of 0.013 mg ethoprop/ℓ for the pink shrimp, Penaeus duorarum. The 48-hour EC_{50} for decreased numbers of normally developing embryos of the eastern oyster, Crassostrea virginica, was 16.0 mg ethoprop/ℓ under static conditions (ERL, 1981).

6.2. CHRONIC STUDIES

Adverse effects from ethoprop were reported in sheepshead minnows exposed during embryonic and juvenile developmental stages (ERL, 1981). A significant ($P<0.05$) decrease in the survival of juvenile fish was reported at concentrations >0.012 mg ethprop/ℓ. Embryo survival was slightly but not significantly reduced at this level of ethoprop. The effects of ethoprop on the growth of juvenile C. variegatus could not be assessed at levels >0.021 mg/ℓ because of poor survival, but ethoprop at 0.012 or 0.021 mg/ℓ had no effect on length of juveniles. No physical deformations were observed among exposed fish (ERL, 1981).

Mysid shrimp exposed for an entire 28-day life cycle showed increased mortality ($P<0.05$) among groups exposed to 0.00062-0.0014 mg ethoprop/ℓ. A significant ($P<0.05$) reduction in the number of offspring produced per female was evident in the group exposed to 0.0014 mg ethoprop/ℓ (ERL, 1981).

6.3. PLANT EFFECTS

The 96-hour EC_{50} for growth reduction in Skeletonema costatum (determined by optical density) was determined to be 8.4 mg ethoprop/ℓ (ERL, 1981).

6.4. RESIDUES

During chronic testing, the BCF for ethoprop was determined in juvenile sheepshead minnows (ERL, 1981). After 28 days of exposure, BCFs ranged from 17 in fish exposed at 0.012 mg/ℓ to 4 in fish exposed at 0.098 mg ethoprop/ℓ. Tissue levels in fish ranged from 0.10-0.48 µg/g and were somewhat dose-related (ERL, 1981).

6.5. OTHER RELEVANT INFORMATION

Additional data relevant to the aquatic toxicity of ethoprop were not located in the available literature.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

No concentration limit in workplace air for ethoprop has been recommended by the American Conference of Governmental Industrial Hygienists or the National Institute for Occupational Safety and Health.

According to the Code of Federal Regulations (CFR), the tolerance for ethoprop residues on peanuts, sugarcane, potatoes, pineapples, mushrooms, cucumbers, corn, cabbage, beans, bananas, soybeans and sweet potatoes is established at 0.02 ppm (40 CFR 180.262).

According to the Federal Register (FR), the U.S. EPA has established a temporary tolerance for ethoprop residues on grapes at 0.02 ppm (48 FR 31083).

7.2. AQUATIC

Guidelines and standards for the protection of aquatic organisms from the toxic effects of ethoprop were not located in the available literature.

8. RISK ASSESSMENT

Because of the lack of pertinent data, no assessment of the potential carcinogenicity of ethoprop can be made. The absence of published information regarding the effects of subchronic or chronic exposure to the chemical precludes the derivation of an ADI. An apparently unpublished study, summarized in a single sentence by the BCPC (1977), indicates that rats and dogs fed 100 ppm ethoprop in their food for 90 days had depression of cholinesterase levels but no histopathologic effects. This dietary level may therefore correspond to a subchronic NOAEL for these species, but the information provided in the secondary source is inadequate for assessment of the study and derivation of an ADI.

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APPENDIX:

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of:

CA SEARCH (Files 308, 309, 310, 311, 320)
TOXLINE
MEDLINE
RTECS
SCI SEARCH
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
EPCASR
Chemical Industry Notes

These searches were conducted in May, 1983. In addition, hand searches were made of Chemical Abstracts (Collective Indices 7 and 8th), and the following secondary sources were reviewed:

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